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Pneumatosis Cystoides
Intestinorum Hominis

Peptic Ulcer—A Chemical Imbalance

The Anal Papilla

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CONTENTS

Features

181 Pneumatosis Cystoides Intestinorum Hominis
Jacob Reichert, M.D., F.I.A.P.

198 Peptic Ulcer—A Chemical Imbalance
Ejnar Jarlov, M.D.
Erik Friis, M.D.
Jacob M. Leavitt, M.D.
Charles R. Dugan, M.D.
Vojslav I. Mitrovic, M.D.
Norman A. Samuels, M.D.
Joseph H. Tuchman, M.D.

206 The Anal Papilla
Thomas R. Peyton, M.D.

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CONTENTS

Editorial	**209**	Prophylactic Proctology
Convention News	**210**	Announcement of Twelfth Annual Convention—1960
Departments	**172**	Letters to the Editor
	213	Book Reviews Textboook of Surgery Ciba Foundation Colloquia on Endocrinology Tumors of the Bladder A Short Practice of Surgery
	216	Newer Medicinals

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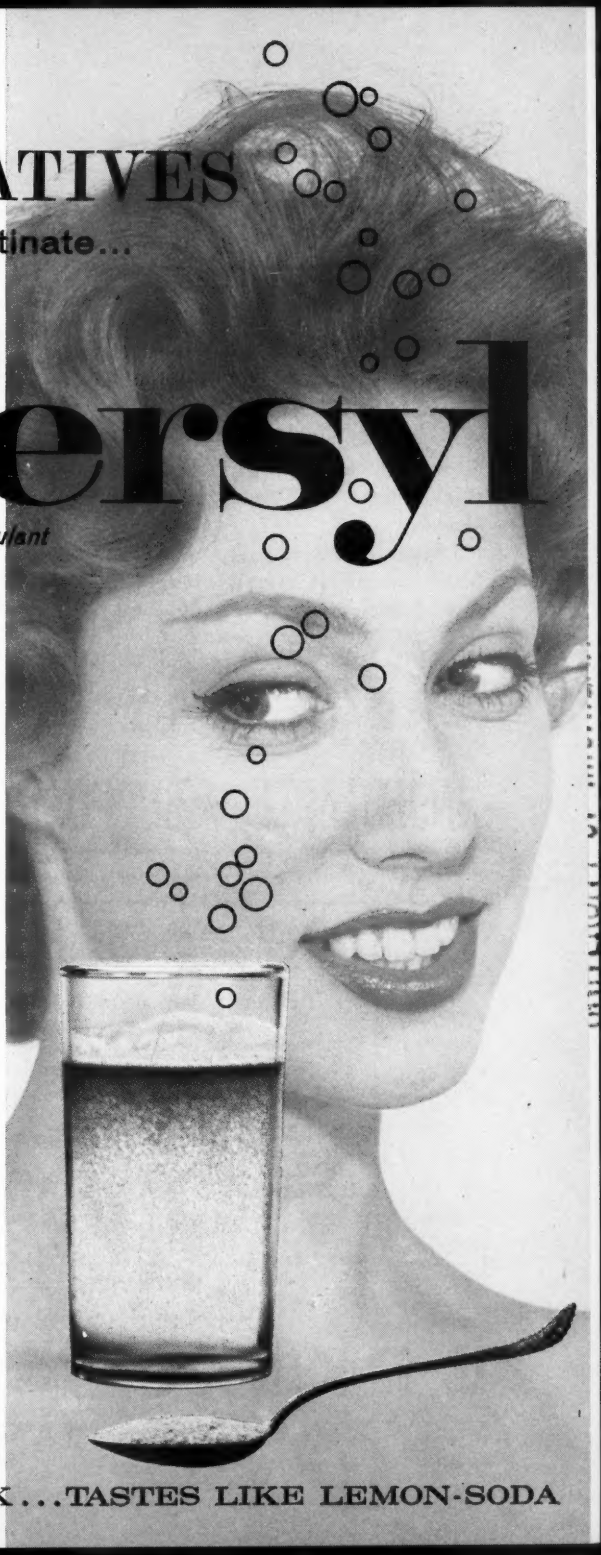

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LETTERS

To The Editor

Dear Sir:

I would like to express my appreciation to the International Academy of Proctology for a wonderful teaching seminar. The panel on psychosomatic medicine was particularly good, and I enjoyed the special evening session on hypnosis.

I hope that we will have more of both when you meet again. Where will the next meeting be, and will there be a registration fee?

Cordially,
RL., M.D., Texas

The 1960 Teaching Seminar of the Academy will be in Miami Beach, Florida, at the American Hotel, April 23rd through April 28th, 1960. There will be much more hypnosis.

The practical applications of hypnosis in proctology, general surgery and medicine, will all be fully developed at that time.

As usual, there will be no registration fee for the Convention. There may, however, be a fee for the hypnosis day, if a special day is set aside for this presentation.

We will look forward to having you with us once again in 1960.

This department is offered as an Open Forum for the discussion of topical medical issues. All letters must be signed. However, to protect the identity of writers who are invited to comment on controversial subjects, names will be omitted when requested.

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Pneumatosis Cystoides Intestinorum Hominis

WITH A CASE REPORT*

The title of this presentation "pneumatosis cystoides intestinorum hominis" will be interchanged often with the following terms: pneumatosis, pneumatosis intestinalis, intestinal pneumatosis, blebs, blisters, vesicles, gas cysts, intestinal gas cysts, intestinal blebs, intestinal vesicles.

JACOB REICHERT, M.D., F.I.A.P.
Phoenix, Arizona

There are no barriers to duration, distance, notion, thought and time, when medical research and knowledge depends upon it.

The attainments in science do not denote the time or age of fulfillment. Those engaged in the practice of science not only admit that "Age cannot wither her," but to the contrary, feel that "Age, I make light of it, Fear not the sight of it."

The appellation of "Pneumatosis Cystoides Intestinorum Hominis" tries to depict an established anatomical entity which is the result of a rare intestinal disease. Seldom does such a short heading of a topic describe so much. However, as far as the etiology and pathogenesis of pneumatosis is concerned, it must be admitted that our present knowledge and armamentarium has not increased proportionally with the course of time.

Pneumatosis Cystoides Intestinorum Hominis is a rare disease and the first two orderly descriptions were reported by Du Vernoi and by F. De P. Combalusier in 1747 during an autopsy. Since so long a time, approximately 230 cases have been reported in the medical literature.

Until 1956, 49 cases were authenticated in the United States, of which just a few were diagnosed preoperatively, some were found while operating, and the rest were observed during autopsies.

Patients afflicted with this disease are extremely emaciated and bedridden for a number of years due to its chronic nature. It may be affirmed that pneumatosis seldom, if ever, appears in an acute episode at the early stage of the disease. Because of its chronicity, slow progress and progressive changes of personality, the average person does not seek medical attention. It is usually the patient's family that becomes aware of the gradual deterioration, and the proper medical attention is then

* From the Department of Surgery of Memorial Hospital, Phoenix, Arizona.

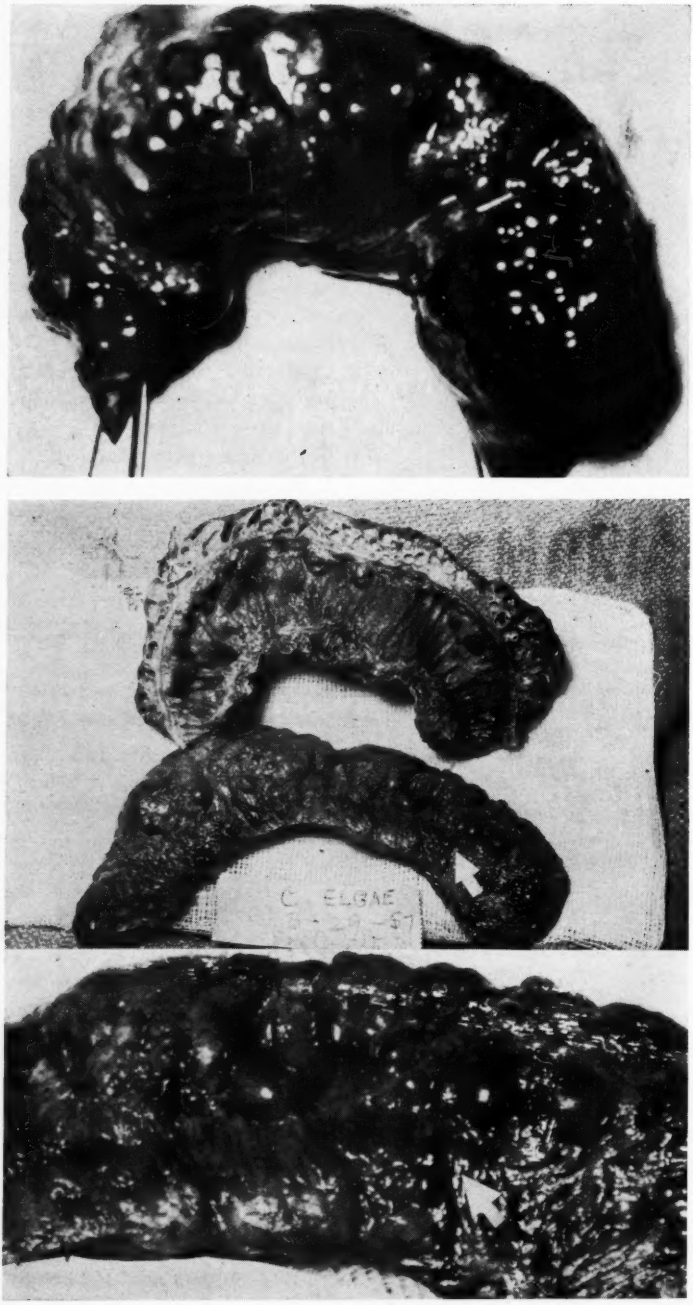

Fig. 1. Fresh resected surgical specimen of terminal ileum, photographed three hours after its removal, revealing innumerable gas cysts and granuloma-like formation.

Fig. 2. Preserved opened specimen, Formalin-fixed, showing unusual thickness of serosa containing innumerable honey-combed spaces of various size and shape; these empty spaces were filled with a gaseous substance, forming the described blebs or gas cysts. This specimen is split along its longitudinal axis and shows the serosal and mucosal surfaces of the removed small intestine.
a. The mucosal aspect reveals protruding cysts through its lining of which many were somewhat pedunculated and most of them having a large base. b. The serosal surface of the specimen, despite its long stay in formalin and the resulting shrinking, still visualizes the granuloma-like formations, gas cysts and the restricted part which intussuscepted. c. Arrow indicates the area of enlargement: visualizing circumferences of existing gas cysts.

provided; such decision often coincides with the symptoms of an acute intestinal obstruction.

Seemingly slow progressive inanition and the resulting involvement of the gastrointestinal tract, establishes a peculiar apathetic behavior.

The evoked indifference and drastic changes of personality in those who are chronically affected with Intestinal Pneumatosis is likewise characteristic and of diagnostic importance in paralytic ileus, and intestinal obstruction if not even associated with this pathologic entity.

In reviewing the past and present literature, the impression remains that the incidence of Pneumatosis afflicts mostly the middle age group of the male population, followed by females and children.

Cystic Pneumatosis was seldom described as a primary affection, due to its unknown pathogenesis and variable syndrome.

The most frequent recorded findings including those in the presented case, are the almost constant association with chronic disturbances of the G.I. tract. The most common pathology encountered is pyloric stenosis, gastric and duodenal ulcers, chronic appendicular infection and progressive intestinal obstruction. However, any chronic morbid affection of the alimentary tract, regardless of its location, may predispose to the formation of intestinal gas cysts.

Main Features of Pneumatosis Cystoides Intestinorum The characteristics of pneumatosis intestinalis are formations of innumerable blister-like blebs under the visceral serosa along the intestinal tract. These blisters vary in shape and size and are filled with gases under tension. These cyst-like formations closely resemble foamy mountains of detergent bubbles which are transparent and quite resistant to pressure. Each cyst, regardless of its size, is separated from the adjacent one by a very thin septum. Most of the cysts are rounded and are separate units hermetically closed. The film-like membranes covering the gaseous cysts are

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Presented at the Tenth Annual Teaching Seminar of the International Academy of Proctology, Mexico City, Mexico, April 9-13, 1958.

shiny, clear, translucent and colorless (See Fig. 1).

The red reflection of the cysts mirrors the compressed septums and the cystic floors and can be compared to the cornea which reveals, through its transparency, the color of the eyes.

Another peculiar characteristic of intestinal pneumatosis is the unusual thickness of the visceral serosa and the continuous reproduction of granuloma-like protuberances within the serosa which often assume the size of an orange or grapefruit. These large meaty masses are the result of multiple overlapping air cysts which resemble grape clusters, and the increasing thickness of the serosa is due to interstitial cementing connective tissue reaction (See Fig. 2).

During the active stage of intestinal pneumatosis there is almost always free air present underneath the diaphragms, and a certain amount of ascites is present in the abdominal cavity. The peculiarity of this transudate which has never been reported in the literature up to date, is its particular viscosity, which is oily, foamy, frothy, exceedingly slippery and can be compared to slightly agitated liquid detergent. Strangely enough, despite the involvement of large areas of the intestinal tract and the hypertrophic reaction of the visceral serosa, the formation of adhesions is minimal.

Intestinal obstructions, intussusception and volvulus are the usual complications of pneumatosis. Pronounced abdominal distention and meteorismus is usually present during the active stage of the disease.

Before any further discussion of the etiology, pathogenesis, and a review of the existing theories is presented, a typical case of pneumatosis cystoides intestinalis hominis will be described.

Case Report Mrs. C. E., a white female age 65, was seen by me for the first time March 8, 1957.*

The main complaints were: severe nausea and vomiting, inability to retain fluids or solids, abdominal distention and weakness.

HISTORY: Family history was non-contributory. The patient was married and childless.

Past history reveals an operation for a ruptured appendix in 1942, and a complete hysterectomy and a segmental resection of six inches of large bowel in 1945.

According to the patient, she never completely recovered from the last surgery and has remained in a weakened condition with periodic vomiting and gradual loss of weight.

From 1946 to 1953 the patient was hospitalized for pneumonia a number of times, and since then she has become progressively weaker; developing pain in the R. U. Q. followed by frequent vomiting, chills and elevated temperature. Intermittent episodes of abdominal distention and obstipation accompanied the above condition. Subsequently she was hospitalized October 14, 1953 and extensive x-ray and lab studies were made. The x-ray findings revealed non-visualization of the gallbladder and a pneumoperitoneum with the presence of free air under both diaphragms (See Fig. 3).

---

* This patient was seen in consultation at the request of Robert M. Stump, M.D., to whom I am very grateful indeed, and it is a privilege to have had the opportunity of describing this interesting case and to review the entire history of this disease from its inception.

Fig. 3 (top). Roentgenogram taken October 16, 1953, shows free air under both diaphragms separating the liver and spleen from the parietal peritoneum. The original film reveals a foamy bubble like surface on the rounded part of the liver, resembling cystic formations. It is probable that slipping of the small bowel under the diaphragm took place showing the serosal cysts or cystic formations on the visceral serosal of the liver itself.

Fig. 4 (bottom). Roentgenogram taken February 15, 1957, reveals tension peritoneum with severe displacement of the liver medially and downward. The original film reveals also the effects (a). of the endoabdominal pressure on the liver-vascular-pedicle and explains the formation of a "caput medusae" like collateral circulation appearing on the abdominal surface as mentioned in the text. (b). The same film shows also the exact outline of the left kidney and complete displacement of the spleen resting almost in the pelvis. The small bowel is enormously distended with definite evidence of fluid levels and paralytic ileus.

The possibility of a hollow viscus perforation was considered. However, additional studies made a few days later could not demonstrate nor locate the above lesion, but greater tenderness became evident and more accentuated in the R. U. Q.

Due to the persistence of obstipation, distension and symptoms of intestinal obstruction the diagnosis of "Cholecystoduodenal fistula with impacted gallstones in the ileocecal valve and intermittent intestinal obstruction" was made. A laparotomy was performed October 23, 1953 and the preoperative diagnosis was not confirmed. The essential findings during the exploration were: "the gallbladder was greatly distended . . . the cystic duct was extremely small . . . the serosal surface of

the entire abdominal contents had the appearance of reaction as to a perforated hollow viscus such as a peptic ulcer . . . the small bowel was run throughout its length . . . except for the serosal reaction described there was no sign of any perforation, and no other lesion." The gallbladder was removed.

The pathological report indicated that "marked chronic suppurative cholecystitis" was present.

From 1953 to 1957, the patient was seriously ill at frequent intervals, however, no detailed information was available.

The next hospital admission was February 15, 1957 with a diagnosis of "possible bowel obstruction." This readmission was occasioned by the gradual increase of abdominal distension and severe pain in the epigastric region for five days prior to admittance. Roentgenologic studies made by Dr. John W. Kennedy indicated that: "Abdomen: Flat plate of the abdomen shows massive amount of air under either diaphragm and it rotates out in the flank on the lateral decubitus, and there are also fluid levels in the small bowel pattern, which is markedly distended (See Fig. 4).

Now going back to films in 1953, she had free air under either diaphragm at that time, but not so much distention of the bowel pattern, although it was considerably distended. Now we either have to suppose that she has ruptured a viscus and that she has escape of free air into the peritoneal cavity with paralytic ileus *or a rare condition but which probably obtains in this case, and that is an accumulation of air cysts in the mesentery of the small bowel which rupture, and give this free air in the peritoneal cavity. This would be more likely since it happened on two occasions.* But now she is in real trouble with evidence of bowel obstruction because of the fluid level in the small bowel pattern.

CONCLUSIONS:

1. Probably serious cystic disease of the mesentery, the rupture of cysts.
2. Paralytic ileus.
3. Bowel obstruction?"

Note: Reviewing the x-rays of February 1957, it is definitely evident that the diagnosis of "Pneumatosis Cystoides Intestinorum Hominis" was established by Dr. Kennedy. However, the cystic formations of the intestine were not mentioned due, probably, to the simultaneous presence of gas cysts on the intestine and mesentery, overlapping each other and easily exchanged.

The patient was treated conservatively and discharged from the hospital after 19 days with no improvement.

Physical Examination Examining the patient in consultation March 3, 1957, she was observed to be a severely emaciated 65 year-old white female evidently suffering from malnutrition, who appeared seriously ill. It is noteworthy to mention the patient's great indifference and the predominance of a calm, tranquil and composed attitude with a monotonous eloquence, assuming at times a witty role; this unusual burlesque behavior without emotional participation is usually observed in those afflicted with chronic peritonitis and progressive intestinal obstruction.

The skin was undernourished, loose and wrinkled, with a subicteric, ashy color as seen in chronic debilitating metabolic diseases of long duration due to prolonged starvation. The chest was very emaciated with severe atrophy of all

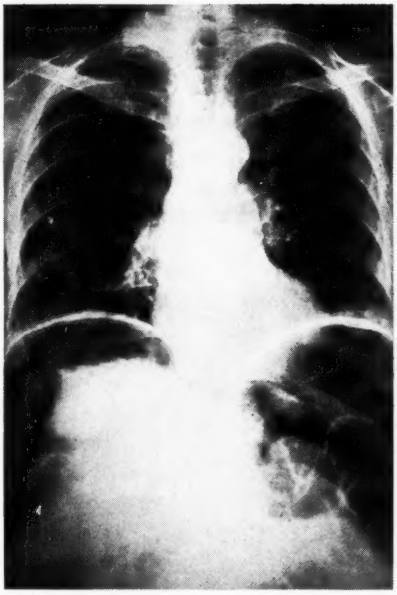

Fig. 5. X-ray taken February 22, 1957, of the chest and upper abdomen shows still pronounced pneumoperitoneum with some atelectasis at either base due to high position of the diaphragms. There is still dilatation of the small bowel pattern, but less marked than in February 15, 1957.

integuments and underlying structures; there was complete atrophy of the mammary glands.

Auscultation revealed minimal and shallow breathing, however, dyspnea was not apparent. Heart sounds were active, efficient, regular and rhythmic. The valves were intact with no leakage. The B.P. in both arms was 130/85 with no fluctuations during the respiratory phases.

The abdomen was distended with intermittent pronounced tension, pliable and covered with a very thin skin, revealing collateral circulation resembling somewhat an established by-pass observed in liver cirrhosis with the formation of a "caput medusae." Peristaltic waves of distended bowel, appearing at irregular intervals, were noticeable as seen in congenital megacolon.

While laying on her back with flexed thighs and legs (seemingly her most comfortable resting position) palpation of the abdomen elicited accentuated pain in the R. U. Q.; percussion revealed pronounced tympanism which included the entire hepatic region, indicating displacement of the liver. Auscultation revealed rushes of loud borborygmi rather than the usual peristaltic sounds; however, its intensity was less than would be expected. Assuming a lateral position, severe distress was elicited in the opposite side and disclosed the presence of a fluid level in the abdominal cavity.

Patient was readmitted to the hospital March 11, 1957, and extensive blood chemistries and x-ray studies were ordered. The chemistries disclosed severe dehydration, depletion of electrolytes and an abnormal A/G ratio. The x-ray films showed pronounced pneumoperitoneum, highly situated diaphragms, pronounced distension of the small and large bowel with levels of stepping fluid. The presence of honeycomb-like formations were noted suggesting multiple gas cysts of the intestinal wall. The very slow and prolonged process of the barium meal indicated incomplete obstruction of the bowel (See Fig. 5).

As soon as the above condition was established decompression was started with intermittent suction, leaving between times, the free end of the Miller-Abbott tube attached to a higher level of the bed, above the head, to act as an exhaust. The general dibilitated condition of the patient gradually improved by correcting the abnormal blood chem-

istries, dehydration and the A/G ratio; however, regardless of all this, the inability to retain fluids or solids continued, and it became evident that a complete intestinal obstruction was present. A laparotomy was decided upon and sterilization of the intestine was attempted with a solution of Neomycin through the Miller-Abbott tube and enemas q. four hours, the day before surgery.

The operation was performed using a high spinal anesthesia on March 30, 1957, and the pathological findings were of unusual interest.

Exploratory Findings In opening the peritoneum the sensation of a great amount of escaping air was noted. The texture of the peritoneum was hypertrophic, fibrous and of greater thickness than the abdominal fascia.

Inspection of the viscera prior to any displacement revealed the intestines to be completely immersed in a 2000 to 3000 cc. pool of foamy liquid. This fluid was clear, transparent and probably of transudative origin; the viscosity was glycerine-like and slippery and the entire liquid pool was covered with a layer of foaming bubbles simulating a slightly agitated liquid detergent.

Further inspection disclosed that the small intestine was literally covered with innumerable growth-like foamy formations of gas cysts, varying in size from a pinhead to a pea (See Fig. 6).

The small intestine was then progressively exteriorized and examined. It was noted that the entire visceral serosa contained an infinite number of blisters separated from each other, in a honeycomb fashion. The distribution of the cysts was irregular and in certain areas the multiplication was enormous, overlapping each other, and forming, at times, a cauliflower-like configuration, assuming the characteristics of large granulomas. About 15 to 20 inches above the junction of the ileocecal region, there was a granuloma-like formation about 12 inches in length involving the entire circumference of the small bowel. This particular growth contained ballooning protuberances and seemed to be the site of the obstruction. Palpation of this meaty mass revealed it to be intussuscepted in its distal, dilated segment. Disengagement was attempted to obtain patency however, the size and length of the affected area did not permit maneuvering without exposing the remaining parts to excessive trauma. The distension of the small bowel above, and the collapsed part underneath the intussuscepted area, made it easy to locate the site of the obstruction. Occasionally it was found that parts of the mesentery were also involved, however, the jejunum, duodenum, stomach and large bowel were not affected.

It may be mentioned that the entire length of the intestinum crassum was found to be collapsed, contracted, and empty of matter and air, contrary to the observed distension on multiple films.

The liver was small, with sharp edges, of tobacco leaf color, and hard in consistency; the spleen, however, was enlarged. The stomach was about three times its normal size and constricted near its antrum, assuming the form of an hourglass. The gastro-pyloro-duodenal region was carefully examined, the passage was found restricted but not obstructed. The duodenum and head of the pancreas were normal, however the entire hepato-duodenal ligament and gall-bladder area were involved in

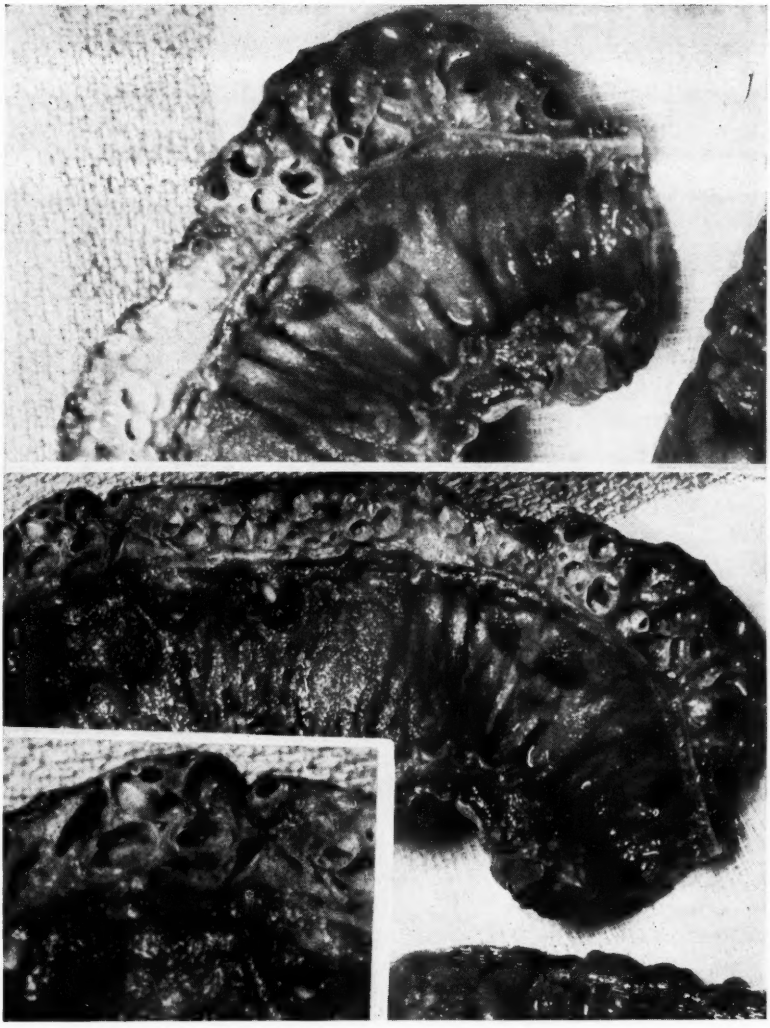

Fig. 6. These illustrations and insert are croppings of Fig. 2, to demonstrate clearly the thickness of the serosa, the protruding submucous cysts and the honeycombed configuration of the cystic spaces.

a mass of adhesions. The uterus had been removed during a previous operation but atrophied ovaries and tubes were present. The urinary bladder was found to be relatively small.

Completing the exploration, a segmental resection of the obstructed part of the small bowel was performed. The postoperative course was practically uneventful. The only transitory complication occurred the second postoperative day, when it was noted that a certain amount of subcutaneous emphysema was present in both inguinal regions and to a lesser extent in the flanks. In removing the dressing it was noted, moderate emphysema was also present in the incisional area due, probably, to leakage of air from ruptured intestinal blebs. Further prevention of such infiltration was accomplished by applying a pressure dressing and within a period of ten days no further emphysema was noted. A scout film confirmed the absence of free air in the abdomen and interpreted as the cessation of formation of gaseous substances.

Pathologic Report Macroscopic: The description of the serosal coverings of the small bowel, as reported by the pathologist, reveals that its entire surface was very irregular and presented numerous small cystic structures measuring from a pinpoint to 10mm. in diameter. On the cut surface, the subserosal area measured 1½ cm. in width and presented a honeycomb appearance as a result of the presence of numerous small cysts (See Fig. 7).

The mucosa for the most part was intact, presenting transverse ridges with an occasional small cyst projecting through its lining. Most of the blebs contained a gaseous substance.

Microscopic: The microscopic findings of the small bowel mucosa showed a typical intact lining of the epithelium and normal appearing glands. The submucosa was congested, containing dilated vessels, and occasionally large cystic spaces lined by a flattened layer of epitheloid cells were noted; their presence in the submucosa was evidenced by a wart-like mucosal elevation, visible to the naked eye (See Figs. 8, 9, 10).

The muscularis and the muscularis-muscosae appeared to be normal. Foreign body type monocytic cells, multinucleated in some areas within the submucosa, were scattered. The majority of cysts were located in the grossly thickened serosa layer. Adjacent tissues showed evidence of active fibrosis, with replacement of fat, combined with many chronic inflammatory cells. No specific inflammatory reaction nor acute inflammation was noticed. Guinea pig inoculation of the transudate and cultures on Sabouraud's and Littman's oxgeal, were negative.

Patient continued to improve, her intake was increased gradually and a weight gain of five pounds was noted in two weeks. Small amounts of refined psyllium seeds with adequate hydration was added daily to the low residue diet to establish intraluminal filling of the intestine, in addition to small amounts of cholinergic drugs to stimulate and promote peristalsis.

For the patient's convenience the continuance of the hospital stay was extended and permission for daytime outings and home visitation with her family was permitted. For a period of six weeks she continued to do well, increasing appetite, greater intake of aliments, and improving disposition and cheerfulness.

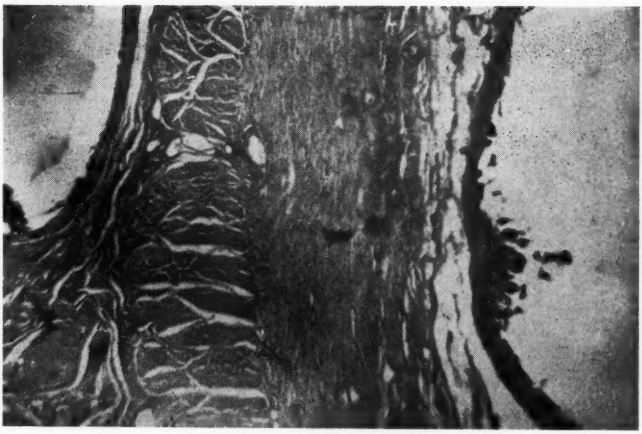

Fig. 7. Photomicrograph of small bowel —surgical specimen. Submucosal and serosal cysts with intervening muscle layer. X20.

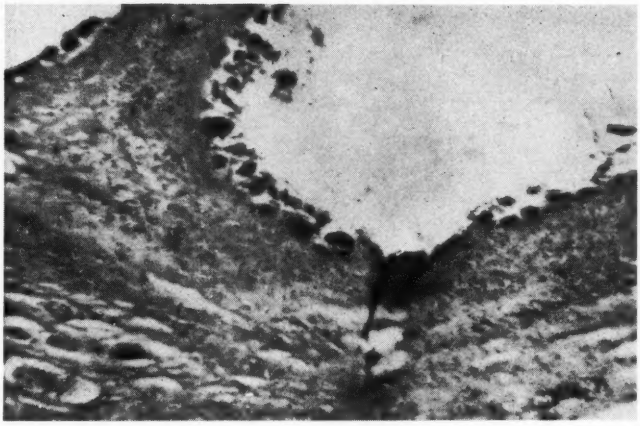

Fig. 8. Photomicrograph of small bowel —surgical specimen. Wall of cyst showing prominent lining cells. X55.

Acknowledgement is made to George Scharf, M.D. for the courtesy of preparing all microphotographs and their respective legends.

The beginning of June, postprandial nausea, followed by vomiting, was reported by her husband; however, this was brought to our attention two weeks after its onset. This information was withheld from us due to the patient's unwillingness to discontinue her outings.

A scout film of the abdomen disclosed extremely dilated small loops located particularly in the R. U. Q. The patient became seriously ill with increased vomiting and abdominal distension. Suction was applied and it was noted that the gastric tube did not advance beyond the jejunum.

It was evident that the newly formed obstruction should be surgically relieved; however, the operative permission was withheld for some time, until the patient's condition became extremely critical and there was no alternative.

Findings of the Second Laparotomy July 17, a laparotomy was performed under local anesthesia and the obstruction found in the R. U. Q. was

Fig. 9. Photomicrograph of small bowel —surgical specimen. Submucosal cysts with prominent lining cells. X20.

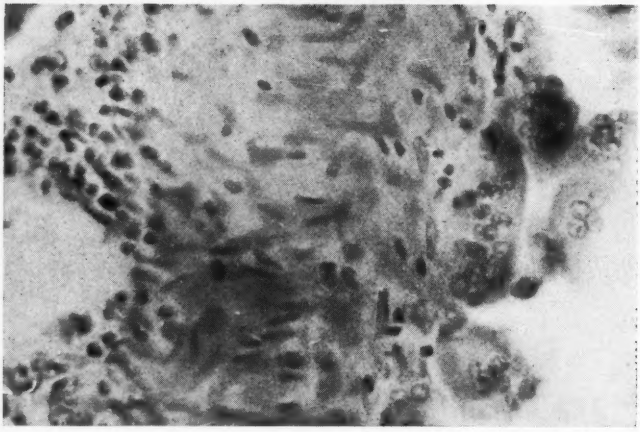

Fig. 10. Photomicrograph of small bowel —surgical specimen. Deep portion of mucosal glands with portion of submucosal cyst wall. Note multinucleated giant cell of "foreign-body" type. X245.

an intricated volvulus of the jejunum and duodenum, forming several solid knots.

To our great astonishment it was found that the process of pneumatosis cystoides intestinalis was completely arrested and no part of the intestinal tract contained gas cysts. X-rays taken prior to the operation revealed the absence of gas cysts and disappearance of pneumoperitoneum; nevertheless, to see it in vivo was quite impressive.

The entire length of the colon and small bowel was examined and found to be patent and the site of the previous segmental resection was completely healed.

Disengagement of the volvulus was easily accomplished due to the recent, loose, inflammatory adhesions.

Notwithstanding the short procedure, the patient's condition remained critical, comatose, and with no bodily movements nor reflexes present. It was the impression from a number of examinations made, that a C. V. A. prob-

Fig. 11. This illustration shows stenotic area and hour glass formation of the antral area of the stomach. The same stenosis with distended stomach can be seen in Fig. 15, before displacing the abdominal viscera during necroscopy.

Fig. 12 (below). Small bowel with all visceral abdominal organs separated from body on board, showing the twisted ligament of Treitz, discolored ansa of afferent jejunum with its twisted mesentery, which reveals to be edematous and partially necrotic where the strangulation took place. This illustration does not reveal the presence of gas cyst, which were affecting the entire length of the small bowel in March 1957.

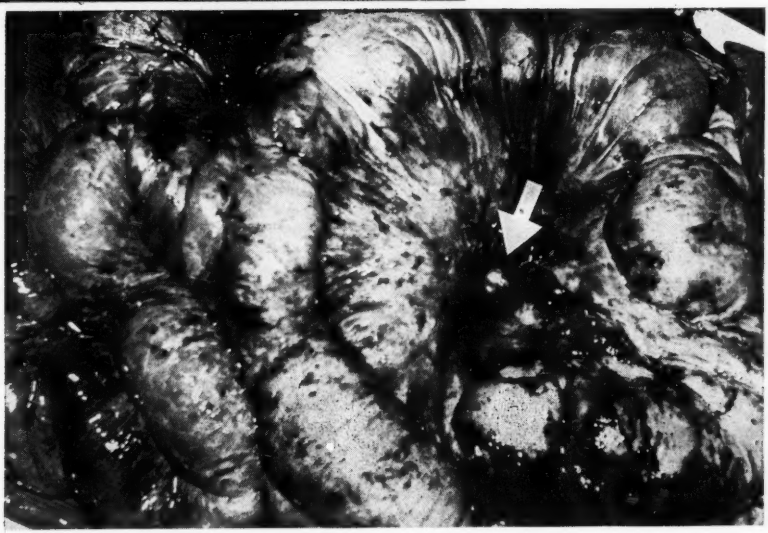

Fig. 13. Part of the small bowel where the "end to end" anastomosis was performed March 1957, revealing complete healing with no intermediate scar tissue, stenosis or granulation tissue. The mucosa was intact and the intestinal lumen was patent. Same condition can be seen in Fig. 17.

ably took place before or during the operation, and she expired fourteen hours later.

An autopsy was performed July 18, 1957. The postmortem findings were of clinical and surgical interest and will be briefly summarized:

Macroscopic: The stomach was three times its normal size with a pronounced stenosis in the preantral portion, assuming the shape of an hourglass. The presence of abundant scar tissue, on both anterior and posterior surfaces of the stomach and adjoining structures was noted *(See Fig. 11)*.

The intestinum tenue was distended and markedly discolored just below the ligament of Treitz, involving the afferent loop of the jejunum. The jejunum and the mesenteric attachment were twisted, edematous, purple in color, and filled with gas and liquid. There was no evidence of gas cysts at the time of the autopsy *(See Fig. 12)*.

The anastomosed terminal ileum, from the previous operation, disclosed an intact mucosa with no evidence of scar tissue and no signs of obstruction *(See Fig. 13)*.

Conclusions of Gross Anatomical Diagnosis The gross anatomical diagnosis was: Acute ileus, strangulation of the upper jejunal loop, multiple adhesions of the peritoneal cavity, pleurae, pericardium, nephro-atherosclerosis, greatly dilated stomach with presence of extensive scaring, causing an hourglass configuration.

Microscopic: Several sections of the intestinal mucosa did not show unusual epithelium. The underlying submucosa and muscularis revealed evidence of severe congestion and edema. The peritoneal surface was covered with a thick organized exudate containing many fibroblasts, blood vessels, and collections of acute and chronic inflammatory cells. There was no microscopic

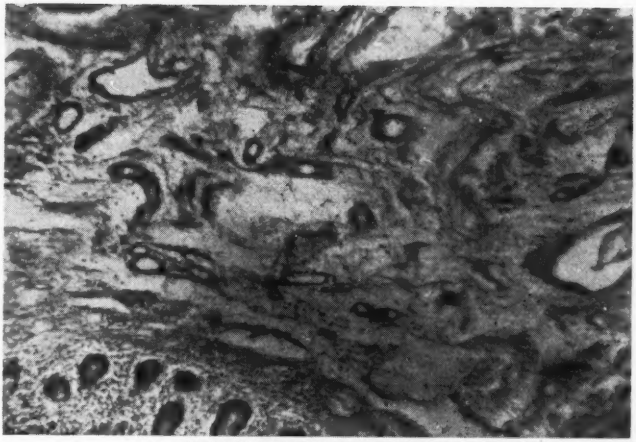

Fig. 14. Photomicrograph of small bowel —autopsy specimen. Mucosa and submucosa showing edema but no cysts. X55.

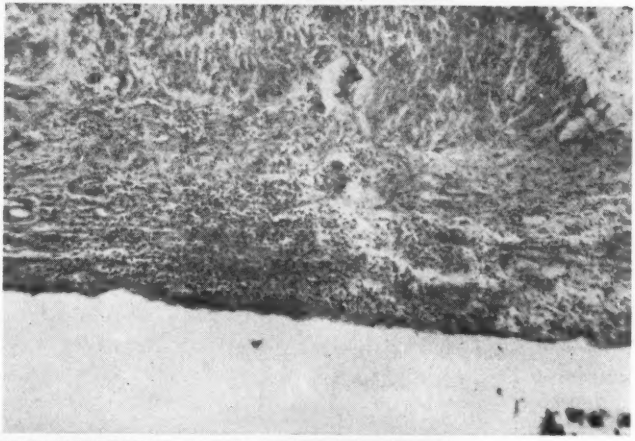

Fig. 15. Photomicrograph of small bowel —autopsy specimen. Muscle and serosa. Inflammation and no cysts. X55.

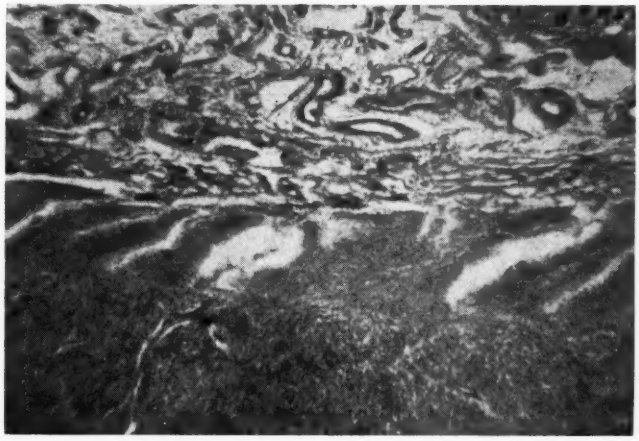

Fig. 16. Photomicrograph of small bowel —autopsy specimen. Complete absence of cysts. X20.

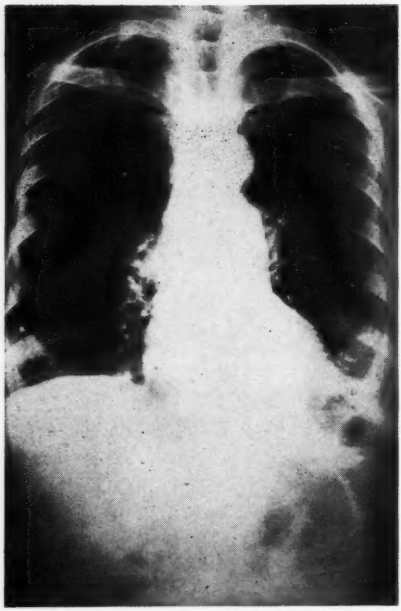

Fig. 17. The chest film dated May 23, 1957 reveals complete cessation of air forming gas cyst with no pneumoperitoneum and no air under either diaphragm.

evidence of gas-cysts in the intestinal walls *(See Figs. 14, 15, 16)*.

The stenotic portion of the stomach did not disclose evidence of previous ulcers or any other lesion of significance on the mucosal layer, however, no muscular fibers were present in the constricted region.

The epicardium appeared irregular and covered in part with fibrin, but with no inflammatory changes.

The pericardial adhesions were dense and fibrous.

The spleen revealed focal capsular thickening with organized surface exudate and calcification in the deeper strata.

Liver surface showed many fibroblasts, congested vessels, collections of chronic inflammatory cells and partially organized exudate.

Comments Most investigators report the great incidence of pyloric stenosis, chronic duodenal ulcer, intestinal obstruction, or some other form of chronic pathology along the gastrointestinal tract, usually concomitant with pneumatosis cystoides intestinorum. In our case chronic gastric stenosis and intestinal obstruction were present; however, the chronic obstruction in the terminal ileum was removed during the first operation, the partial gastric stenosis remained, and x-rays made fifteen days later substantiated the cessation of gas cysts formation and the absence of free air in the abdomen *(See Fig. 17)*.

The question remains: What was the evoking causative factor for the presence of pneumatosis in our case? The removal of the obstruction seems to coincide with the disappearance of gas cysts and may be attributed to it.

The macro- and microscopic pathologic report disclosed a normal intestinal serosa with no blebs and an intact mucosa with the absence of gas cysts formation; nor was there noted any breakage in its continuity to permit passage of compressed gases from the distended gut.

It is quite possible that the patient's chronic malnutrition with all its implicated repercussions on the basic metabolic processes contributed to changes in the intestinal absorption, osmosis and chemical reactions, causing pronounced circulatory difficulties and interference in the lymphatic intestinal flow.

Acknowledgement is made to Morris' **Human Anatomy**, Ninth Edition, for the Teichmann drawings, P. Blakiston's Son & Co., Inc. and to the McGraw Hill Book Company, Inc., publisher of the 11th Edition.

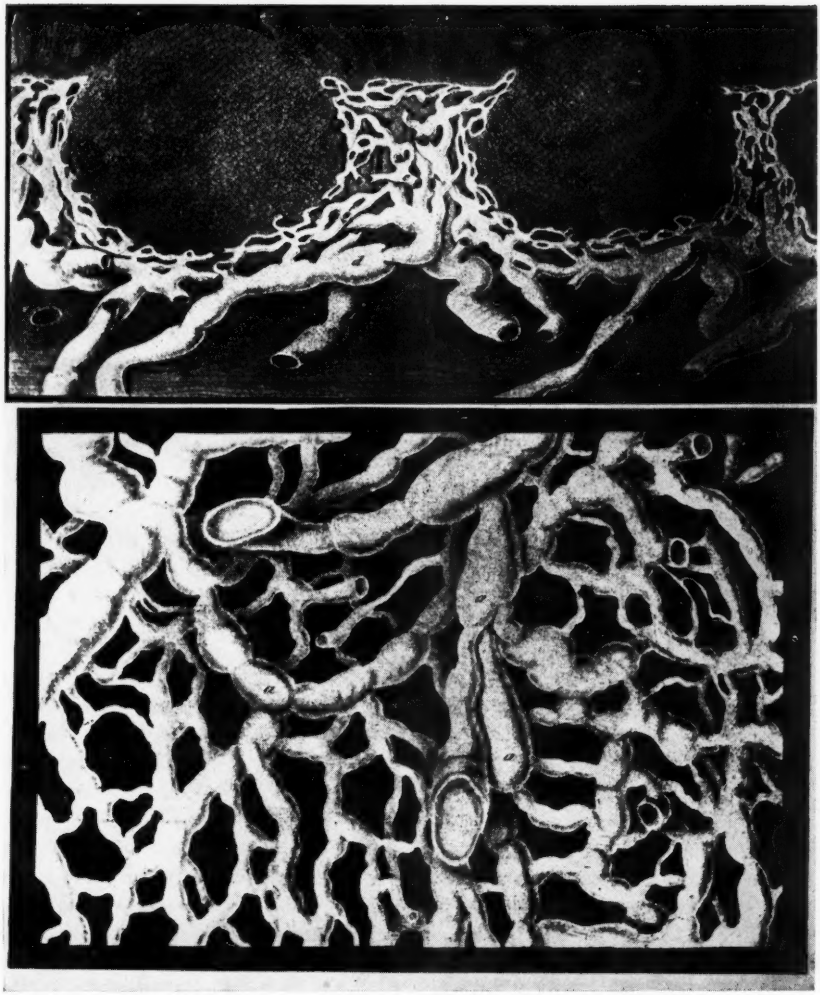

Fig. 18. Surface view of intestinal lymph-nodes and scrotal lymphatics showing typical sausage-like arrangements, corresponding to the distribution of the intestinal cysts in Pneumatosis.

To be concluded next month

Report of a New Therapeutic Approach

PEPTIC ULCER — A Chemical Imbalance

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Medical research and thinking necessarily follow a pattern of timeliness, one might almost say, of fashion. The responsible doctor cannot help but look with suspicion on radical change; only rarely does such change have the tremendous impact of complete and immediate proof as with Pasteur, Ehrlich, Banting and Best.

Pasteur opened a vista of certainty toward which medicine had long been groping. Jenner and Semmelweiss had been frustratingly near. The thousand year old theorizing on the causation of disease was suddenly replaced by a solid structure of laboratory diagnosis, specific serum therapy, antiseptic surgery, and finally the broad spectrum antibiotics.

This concept of disease, as the battleground between a living host and an invasion of living parasites, has so completely overshadowed all medical thought, that it is difficult for us to remember that it was wholly unknown as recently as eighty years ago. Louis Pasteur died sixty-three years ago, and Paul Ehrlich lived to see the First World War, and there are those of us who remember him well.

Other aspects of disease that have been forced from the center of the stage, are beginning again to have importance, as the conquest of the microbes becomes more and more complete. Older ideas of chemical imbalance and of nutritional states, with mysterious symptom pictures, are being re-examined with new interest. We are remembering that Lind saved countless British

sailors from a baffling hemorrhagic death with lime juice almost 200 years before Vitamin C was known.

A recent editorial[1] asks us to take a second look at those diseases whose origin is still unknown. The article praises the work of Tom D. Spies,[2] who says, "Today, germs are not our principal enemy. Our chief medical adversary is what I consider a disturbance of the inner balance of the constituents of our tissues, which are built from and maintained by necessary chemicals in the air we breathe, the water we drink, and the food we eat."

The importance of chemistry pervades even the bacterial arena. Diphtheria was a vicious killer whose weapon was a chemical toxin; diphtheria was conquered by antitoxin, a dead chemical. There is a long list of microbes that follow this pattern:—Tetanus, Botulinus, even Typhoid. The lethal unit in each of these is a chemical toxin which circulates in the blood of the host and injures tissues, far from the site of the invading organisms.

The filterable viruses were long the most elusive of the invading killers. Pasteur, who conquered Rabies, conquered a postulated foe, for he never saw his adversary. Even long after the smaller viruses were isolated, they remained hidden, beyond the range of our microscopes, refusing to be blocked by our finest filters. They seemed not only alive, but cunning and treacherous. A quarter century ago, it was reported[3] in a modest note, that viruses did not have respiratory activity. This suggested that they were not living entities at all. Shortly thereafter, a virus was actually crystallized. From that moment on, it was obvious that a dead chemical could become an active and fatal infecting agent, actually reproducing itself in the body, by what is today a well authenticated chemical process.[4]

As we begin to unfold chemical mysteries, we find that Spies' general concept of a "disturbance of the inner balance of the constituents of our tissues" may be applied in many areas where we have hitherto thought we were completely confident of the etiology and mechanism of disease. How much more important that we should not lose sight of this concept where etiology still remains a puzzle and where therapy has never wandered far from symptomatic relief.

Present Status of Peptic Ulcer

Peptic ulcer is such a problem disease. The World Congress of Gastroenterology[5] had a panel discussion on peptic ulcer. There was so little unanimity of opinion on any point, that a summary was practically impossible. There was the usual implication of the emotional factor; this implication is an all too prevalent tendency to brand as neurotic, anything we have not as yet tagged with a label we can prove.

Since we have no established etiological factor in peptic ulcer, our treatment is symptomatic and empiric. The use of antacids to relieve acid eructation and ulcer pain is an old housewives' remedy; a surgeon in Washington's army rebuked an officer for having succumbed to the "soda habit." Sippy, around 1915, formulated the antacid therapy into a complete regime, based largely on frequent sips of milk, and trying to keep the stomach acids neutralized right 'round the clock. All of our modern therapies, our anticholinergics, and even our surgery, still aim at Sippy's target. But most important, from a physiological viewpoint, our methods are laden

with inconsistencies and leave the therapist, as well as his patient, in an embarrassing dilemma between malady and medicine.

A patient whose basic difficulty is impaired digestion and consequent malnutrition, is given a rigidly restricted diet. This patient, whose symptoms are interwoven with frustration, is ordered to abandon coffee, tobacco, alcohol the condiments that add zest to food, and to curtail work and sports. A patient with an ulcer which must be healed by the building of new tissue, is given medication designed to abolish protein digestion in his stomach; this is an unnatural handicap at the initial source of protein building blocks.

All of these medications, when used in effective dosage, have side actions. The patient exchanges his pain for continued dryness of his mouth and nose, difficulty of vision, and sometimes a rapid pulse and other frightening effects. When desperation leads to surgery, there is no assurance that a new set of symptoms will not replace the old, or that nutritive balance will be restored. For surgery physically removes what chemical therapy had previously tried to neutralize. Peptic ulcer surgery is always an admission of the failure of medical therapy. But unfortunately, in a high percentage of cases, surgery has failures of its own.

Such a non-specific and unphysiologic management of peptic ulcer is forced on us by complete ignorance of the cause of ulcer. Thoughtful clinicians have tried to find a specific underlying lack, either in diet, enzyme or hormone activity, that would serve as a key to handling the disease on a specific basis. Everyone agrees on the necessity of improving the general nutritional picture. And, of course, everyone must deplore those measures which curb symptoms, while they inactivate a digestive process.

The evil effects of the restrictions and classical medications have been spelled out in recent reports[6, 7] but while most clinicians are willing to liberalize diets, few seem inclined to abandon antacids.

The Search for a Specific Factor

A search for specific therapies depends, to a large extent, on being able to reproduce at will a comparable illness in animals. In this way, evaluation of therapies can be done under strict laboratory conditions. Such experimental results are not always transferable to humans, but if we are to have a starting point, the animal experiment in the laboratory is a necessity.

These conditions were met when it was reported that gizzard ulcers in chicks could be produced at will, by dietary means.[8, 9] It was now possible to test dietary factors which could neutralize the ulcer-producing diets. The results of mixtures could be tabulated on a numerical basis.

The first significant success was reported in 1951;[10] the combination of fat-soluble and water-soluble extracts of calf's brain, produced an ulcer-protective factor. This factor could actually be assayed for its protective power against the ulcer-producing diets in chicks. Everything was now ready for the final step, the use of this factor as a therapy in human ulcer.

The immediate results of the human tests were disappointing. However, since the initial factor did not produce a specific protective result in chicks until two extracts were combined, and since human ulcer is doubtless more complex than the gizzard erosions in chicks, it seemed logical that a human protect-

ing-agent might need added supporting elements.

Since the aim of the specific therapy would be the avoidance of alkalis, anticholinergic and sedative drugs, so as to place ulcer management completely on a nutritive basis, the protective factor was added to a complex, but easily reproduced foodstuff, dried milk solids. Thinking that it might be possible to produce a palatable wafer which could substitute temporarily for a complete diet, ferrous gluconate and part of the B complex were added, flavoring extracts, and milk casein as a binder. Moulding this formula into a 20 gram wafer,* it contained 135 calories.

The Therapeutic Test This report concerns the use of the nutritional wafer in 141 proven cases of peptic ulcer, followed for periods varying from 4 years for the earliest cases, down to 6 weeks for the most recent. The cases were all selected because they were objectively proved as to diagnosis, mostly chronic and recurrent, with a well-recorded pattern of response to conventional therapy. In each case this pattern of previous response was used for comparison and control. Dividing the 141 cases into male and female groups, the distribution of each group is shown in the table on following page:

The dosage and regime were not immediately standardized. Since the original aim was to substitute the nutritional wafer for all diet and all medication, ten to twenty wafers a day were used, a total of 1350 to 2700 calories. This was given to most of the early cases, but especially to those who had been resistant to other therapy and to those who had massive hemorrhage. As symptoms came under control, and other items of diet were added, the number of wafers was cut down.

After the first year of experience with this regime, it became obvious that complete substitution of diet was not necessary and that the average course of treatment could be standardized at five wafers daily, spaced out between meals for a period of ten days. After evaluation of response, the course might be repeated for an added ten days. Some patients required from then on, a reduced maintenance dose; others remained completely well with no further medication.

The therapeutic result was not considered favorable unless it was achieved by nutritional wafer alone, without the help of medication or bed rest, without restrictions of tobacco, alcohol or diet. Where previous restrictions had been severe however, the change-over was not sudden. Where drugs and antacids had been used to control symptoms they were trailed off within days, sometimes within hours. Diet was left, usually, to the patient's good sense, though they were warned not to challenge the regime by indulging in excesses, in highly seasoned or heavy foods, or those known to produce distress by their own previous experience.

The entire atmosphere of this regime had an aspect of freedom, in itself a valuable therapeutic agent. This freedom was more than merely the relaxation of irksome restrictions. There was freedom also from the dry throat and palpitation which had made other regimes scarcely tolerable; the freedom to work and to play was an economic advantage, and it left less time for intro-

* Now available commercially as EXUL® from Yorktown Products Corporation, 441 Lexington Avenue, New York 17, New York.

MALES (AGE)		20-30	31-40	41-50	51-60	61-70	Over 71	Totals	
NO. IN EACH AGE GROUP		17	30	32	23	12	1	115	
X-RAY Diagnosis	DUODENAL	15	25	18	11	6	1	76	
	GASTRIC	—	—	3	6	4	—	13	
	COMBINED	1	4	8	5	2	—	20	
	NOT DETERMINED	1	1	3	1	—	—	6	115
Duration of HISTORY	LESS THAN 1 YR.	2	6	3	1	1	—	13	
	1 TO 5 YRS.	10	9	10	7	2	—	38	
	6 TO 10 YRS.	5	9	9	7	3	—	33	
	OVER 10 YRS.	—	4	13	7	6	1	31	115

FEMALES (AGE)		20-30	31-40	41-50	51-60	61-70	Over 71	Totals	
NO. IN EACH AGE GROUP		1	5	6	8	5	1	26	
X-RAY Diagnosis	DUODENAL	1	2	4	5	3	1	16	
	GASTRIC	—	2	2	1	1	—	6	
	COMBINED	—	—	—	1	1	—	2	
	NOT DETERMINED	—	1	—	1	—	—	2	26
Duration of HISTORY	LESS THAN 1 YR.	1	2	—	—	1	—	4	
	1 TO 5 YRS.	—	1	1	2	1	—	5	
	6 TO 10 YRS.	—	2	3	2	2	—	9	
	OVER 10 YRS.	—	—	2	3	3	—	8	26

spective magnification of symptoms. The psychogenic reassurance of the ambulatory regime has an advantage noted by many workers.[6]

The evaluation of the therapeutic result in peptic ulcer is difficult and controversial. There is no universally acknowledged objective yardstick.

Healing by x-ray (the abolition of a crater) was not considered a criterion of success, unless attended by marked clinical improvement. On the other hand, marked clinical improvement, especially in chronic cases, was not always attended by x-ray changes, since a scarred bulb is often a permanent deformity, which of itself, is not responsible for symptoms.

In the last analysis, a favorable result is one that must be judged in each case by each clinician for himself.

It is interesting to note that those cases which were destined for eventual success, usually showed the most rapid

TIME OF FIRST REMISSION OF SYMPTOMS AFTER START OF NUTRITIONAL REGIME

	Less Than 1 Day	1 Day	2 Days	3 Days	4 Days	5 Days	6 Days	1 Wk.	2 Wks.	3 Wks.	Little or No Relief
EVENTUAL SUCCESSES	13	16	36	16	10	12	5	5	1	—	— = 114 cases
EVENTUAL FAILURES	2	2	1	1	1	1	1	2	1	1	14 = 27 cases

response in symptomatic relief, quickly discarding symptomatic medication. The onset of relief may be plotted as above:

Those cases which were not destined for eventual success were ordinarily not partially or even temporarily helped to any great degree. If an ulcer were present on a deficiency basis, and if this nutritional wafer supplied that deficiency, it might well be expected that the reversal of both the symptomatic and the nutritional picture would start immediately. In those ulcer cases which had some other mechanism, the nutritional regime would tend to be a flat failure from the start. Indeed the total experience of this study was just that.

The actual count of percentage of successes is most significant in those cases which have had the longest follow-up. Since the study gained momentum and broadened assistance as it progressed, the cases showing the longest follow-up are in the minority; as the work continues, subsequent reports will equalize the numbers. At the present time, the following table gives the overall evaluation of success:

LENGTH OF FOLLOW-UP AND FINAL EVALUATION OF SUCCESS OR FAILURE

	2 Wks.	3 Wks.	4 Wks.	6 Wks.	7 Wks.	1 Mo.	2 Mos.	3 Mos.	4 Mos.	5 Mos.	6 Mos.	8 Mos.	9 Mos.	1 Yr.	1½ Yrs.	2 Yrs.	2½ Yrs.	3 Yrs.	3½ Yrs.	4 Yrs.	
SUCCESSFUL CASES	7	2	5	5	2	4	7	23	2	1	5	3	3	15	9	7	3	5	5	1	= 114 cases
FAILURES	2	2	2	2	1	2	3	7			1	1		2		1					= 27 cases

The "unsuccessful" group were measured by a rigid and uncompromising criterion, but a word of explanation will temper the percentage. In one analysis of the case records, we separated a group of fourteen patients who were slated for surgery, though nutritionally very poor surgical risks. In

these very sick patients, it was decided, as a last resort, to try the nutritional wafer regime for a short test period. Eleven of these patients were saved from surgery, a very gratifying percentage. However, a more important fact which must be stressed is that the three patients who were taken to surgery as "failures" of this regime, had all *gained weight* and so were better surgical risks. Curiously enough, this single positive fact was present in each of the fourteen cases.

A weight gain was the most general characteristic response of the entire series. Though it varied a good deal, from three to seventeen pounds, the average was about eight pounds for an eight to twelve month observation period. Since these were ambulatory patients, busy at their regular work, and eating a practically normal diet, such a positive nutritional balance in ulcer cases is a very satisfactory result.

The overall percentage of success with this nutritional wafer is at least as good from a statistical standpoint as that of conventional therapy. Looking beyond the statistics, we have patients with immediate relief, gainfully employed, many with a steady gain of weight, remarkably free from recurrence, and during the entire treatment, free from the harassment of side effects and restrictions which are almost as bad as the ulcer symptoms themselves.

From a theoretical point of view, the course of these cases has an interesting connotation for the support of the concept that peptic ulcer is a result of a chemical stress or imbalance, a nutritional deficiency disease not too different from those described by Dr. Spies.[2]

Those cases amenable to the nutritional wafer had symptomatic relief that was unusually immediate and complete, paralleling remarkably the protective pattern of the original DAM factor in chicks. If these results in humans were on a psychosomatic basis, it would still not explain the parallel action in five-day-old chicks.

It was suggested by a colleague that the action of the wafer might be the result of a high glutamine content. However, a careful analysis of the wafer showed that it contained practically no free glutamine.[11]

Another possible mechanism which was explored was that the content of the wafer itself might have an antacid or buffering action. Each wafer contains 14 grams of milk solids representing approximately 2 ounces of milk. The five-tablet daily dose would equal a little over one glass of milk per day. This could hardly be considered adequate buffer on which to base successful therapy.

Gastric analyses were performed on a small number of patients, first fasting, then at varied intervals after the ingestion of a wafer. The low pH rose *slightly* and then returned to the fasting level within sixty minutes. Patients who started with almost neutral gastric juice in a fasting stomach, actually had a more acid content within forty-five minutes after the ingestion of a wafer. These results would suggest that the wafers have no particularly significant antacid effect.[12]

Discussion On the evidence presented, it seems inescapable that we believe for the present that these wafers provide some needed factor in the diet, the absence of which causes peptic ulcer in humans, in about eighty percent of all cases.

It may still be argued that a missing

nutritional factor would not regularly produce a lesion in a selected site, but would have a more general pathological effect. Yet all our experience with the deficiency diseases points to specific sites, certainly for onset, like the ulcerated gums in scurvy.

And here again we have another parallel. Sore and bleeding gums are not cured by Vitamin C *UNLESS* sore and bleeding gums in that particular patient are a symptom of Vitamin C deficiency. This could well explain why this nutritional wafer is a flat failure in some cases and an immediate success in others.

If we accept the theory that peptic ulcer may arise on a basis of specific nutritional deficiency, the dramatic cures would occur in such deficiency cases; the flat failures would occur in those ulcers that had arisen on some other mechanism.

Summary

The concept that peptic ulcer results from a specific nutritional deficiency, seems to be supported by the following facts:

1. A specific ulcer-preventive factor, consisting of a fat-soluble plus a water-soluble fraction, has been shown to be measurably effective against chick gizzard erosions.

2. This factor, when incorporated into a wafer with non-specific nutritional ingredients for support, and tested in 141 proven peptic ulcer patients, was measurably effective.

3. The nutritional wafer alone, without the aid of antacid buffers, sedative or anticholinergic drugs, and with little restriction of diet, tobacco, alcohol or activity, gave rapid and complete symptomatic relief, with weight gain and eventual healing to more than 80% of the cases.

4. All cases were ambulatory and remained at work, happily free from the uncomfortable side-effects of traditional ulcer therapy.

5. Relief is either rapid and complete or a flat failure from the start, suggesting specific replacement in those cases where there is specific chemical imbalance or deficiency.

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The Anal Papilla

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It is well known that in early embryonic life the primitive rectum is separated from the proctodeum. There are certain remnants of the ectodermal membrane which are found at the upper end of the anal canal. These are known as anal papillae. These tit-like elevations vary in number from one to six usually. They consist of a small quantity of connective tissue containing nerve fibers, ganglion cells, and sometimes a cartilage or two as well as a blood supply. This area is often referred to as the pectinate line or the anorectal line. (See Figure 1.) The columns of Morgagni caused by

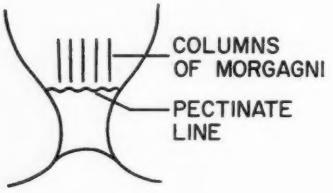

Figure 1

the folds of mucosa above the mucocutaneous junction, end in semi-lunar indentations known as the valves of Morgagni. Behind these are small pockets into which it is possible to introduce a probe to a depth of about one-half inch. These are the crypts often referred to, and may be thought of as the "seat" of infection in this area. There are those who make quite an issue of the excision of these crypts, or cryptectomy, which may seem to attach too great importance to them per se. But these innocent-looking and anatomically accepted protrusions may become the forerunner of serious trouble. It is to trace the probable course of the life of this tiny projection that this account is being written. Because, if it were possible to note and evaluate the changes that ensue in this area from time to time, we might be influenced to regard these so-called innocents with more serious intent.

Many of the anorectal complaints start with a change in the anal papillae and their environs. In fact, these papillae may be regarded as providing some of the earliest evidence of pathologic change in the anorectum. They may give rise to—abscess, fistula (of varying proportion), fissure and ulcer, infected hemorrhoids, pruritus ani and pectenosis. Infection from even such remote areas as the teeth and tonsils is said to often lodge in the anal crypt. Cryptitis may cause irritation and fibrosis of the neighboring papillae, with enlargement. The hypertrophied papilla (usually found dorsally) may produce

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symptoms of tenesmus or the patient may complain of difficulty, incomplete stools, or a crawling sensation. (See Figure 2.) The occasional hard stool has been known to pull down the enlarged papilla with resultant tear at its base, and—anal fissure. (See Figure 3.) Although we are accustomed to look at the hypertrophied skin tab with underlying ulcer as the sentinel pile, actually—the papilla is the true sentinel, for it stands guard over this tiny tear at its base long before the fissure has had time to progress and involve the perianal skin to produce the commonly known sentinel pile. (See Figure 4.) The dorsal commissure, because of its lack of muscular support, is more often the site for fissure occurrence. Pressure from above may push the skin before it to produce the so-called sentinel pile. This of course may occur without involvement or injury to the crypt and without papillitis. Too often, however, it is noted that the fissure, sentinel pile, cryptitis and papillitis occur together.

Symptoms complained of at this stage are those of bleeding with stool, pain with and after B.M., and constipation. These may seem to be relieved by the use of suppositories. Some practitioners resort to the "stretching" procedure to relax the sphincter muscle. Nevertheless, even though there may be intermittent periods of superficial healing and relief, the process becomes more involved, sometimes with infection of the neighboring internal hemorrhoids. We now have the quadruple picture of sentinel pile, ulcer, hypertrophied papilla, and infected internal hemorrhoids—all in one quadrant of the anal canal. (See Figure 5). Other quadrants may be affected also. As this condition is permitted to go on, there are neighbor-

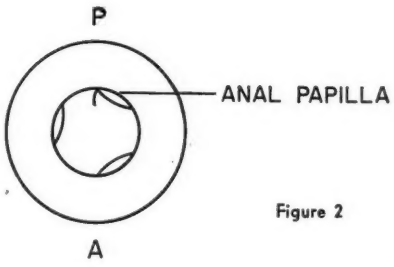

Figure 2

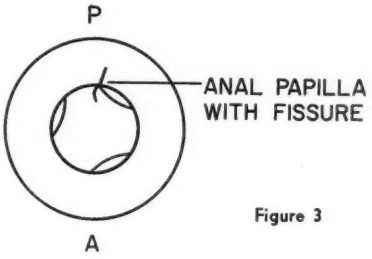

Figure 3

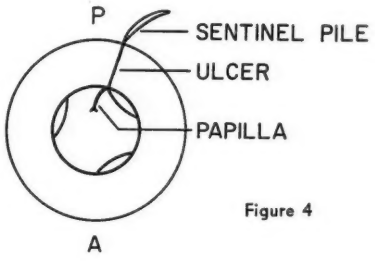

Figure 4

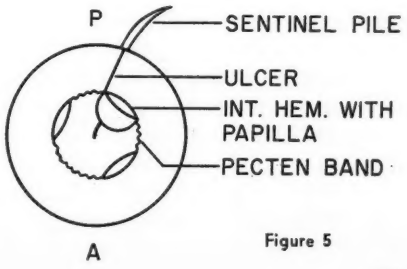

Figure 5

ing problems as pectenosis, edema and swelling of adjacent hemorrhoids, deeper ulceration with involvement of the external sphincter muscles, possibly beginning fistula formation, especially dorsally at the ulcer site, and, of course, abscess formation.

Now we are faced with an anorectal problem deserving the process of surgery as the only means for cure. At surgery it is necessary not only to consider the need for excision of the sentinel pile and underlying ulcer, the enlarged papilla and even the related internal hemorrhoid, but also the division of the pecten band, which by this time is a well developed recognized entity. Some operators unceremoniously incise the subcutaneous external sphincter muscle routinely. This incision usually includes the pecten band. A not too infrequent occasion is the nestling of the dorsal ulcer or fissure between two caressing hemorrhoids at that site, the latter preventing drainage and any attempt at healing. There should also be a thorough investigation as to the depth of infection dorsally. Often a drop or two of yellow pus may be seen to exude from this dorsal site denoting deeper infection before any marked induration or sizeable abscess has had time to develop.

The "lamblike" papilla has now become something of a "lionlike" problem. With careful analysis the papilla may be considered a barometer of sorts for many of the common anorectal disease problems. That early excision of the enlarged papilla may abort later more extreme pathology has been shown to be reasonably possible. This discussion, with the schematic drawings, has been applied to the dorsal site of the anus where this pathology is usually found. Similar or near-similar conditions may prevail in the other quadrants where the papillae are also found. Preventive medicine may find progress by the use of the anoscope for evaluation of the papilla and the adjacent crypts. These problems may be properly appraised with unerring judgment under saddle block anesthesia.

Summary

1. The anal papilla has been described and referred to as subject to serious pathologic change with adjacent involvement.

2. Importance of early diagnosis with emphasis on early changes noted.

3. Delicate surgery as preventive measure in early lesions suggested.

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Resumen

1. La papila anal ha sido descrita y referida como sujeta a cambios patologicos con invasion adyacente.

2. Se hace notar la importancia del diagnostico precoz, indicando los cambios iniciales.

3. Se sugiere la cirujia cuidadosa como medida preventiva en las lesiones iniciales.

EDITORIAL: Prophylactic Proctology

PREVENTION is the better part of therapy. Recent clinical statistical studies have demonstrated that it is possible to prevent a large percentage of lower bowel malignancies. Studies in Cancer Detection Centers, where large numbers of asymptomatic patients are examined, would seem to indicate that 8% of such patients have pre-malignant lower bowel lesions. Obviously, therefore, routine sigmoidoscopy will lead to the early detection of such pre-malignant lesions as polyps, and routine removal—in the office—, will prevent the development of potential subsequent malignancy.

The importance of this one factor in the campaign against cancer cannot be over-emphasized. Hundreds of thousands of lives can be saved annually in this fashion. This is prophylactic proctology at its best.

To a lesser degree in terms of mortality, but to a greater degree in terms of morbidity, abscesses and fistulae can be prevented by prophylactic excision of areas of cryptitis. This is particularly important for the patient who has already suffered an abscess and fistula. It is not enough simply to remove the involved area at the initial time of surgical correction of the acute pathology. It is also essential that a prophylactic cryptectomy be performed at a later date. It should not be performed at the time of the original abscess surgery, for this would simply open avenues for further infection at a time of lowered local and general resistance.

To prevent the development of subsequent fistulae and ulcers, the patient who requires surgery for an anal ulcer or fissure, should, at the same time, be given the advantage of a posterior sphincterotomy. This, combined with the injection of long lasting anesthetics into the sphincter muscles to produce prolonged relaxation, will give the anal outlet sufficient flexibility to prevent subsequent recurrence of fissure or ulcer in the same of other locations.

The best intestinal health—indeed the best gastrointestinal health—, is to be found in those individuals who are especially careful of their nutrition. The French have properly said, "la mort entra par la bouche." Death does indeed enter through the mouth, and most of us dig our graves with our teeth. A properly balanced diet, with a minimum of starch and fat, and a maximum of protein, an adequate supply of minerals and vitamins, and most importantly, low caloric—will improve anyone's nutrition and prevent any gastrointestinal disturbances. The overweight patient should reduce at once. This is the responsibility of every physician, regardless of his specialty. Not only will this prevent or improve many intestinal conditions, but it will help your patients' general health as well.

These are some of the important possibilities of prophylactic proctology. There are others yet to be explored.

It is the duty of the proctologist, not only from the surgical point of view, but also from the general medical and nutritional viewpoints, to prevent intestinal disease as well as to treat it. It is still true that an ounce of prevention is worth a pound of cure.

Alfred J. Cantor, M.D.

The Twelfth Annual

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By popular demand, the International Academy of Proctology is pleased to announce that its 1960 Teaching Seminar will be held in Miami Beach, Florida. The Americana Hotel will be host to the Conventioneers, and the dates will be April 23rd through April 28th, 1960.

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It is suggested that any differences of opinion of individual physicians with any advertisements be called to the attention of the editor, with a duplicate copy of the letter to the pharmaceutical house whose advertisement is the subject of the letter.

THE PUBLISHERS

BOOK REVIEWS

"TEXTBOOK OF SURGERY" by Fred Moseley, Editor, M.A., D.M., M.Ch. (Oxon), F.A.C.S., F.R.C.S., (Eng.), F.R.C.S. (Canada); Assistant Professor of Surgery, McGill University: Associate Surgeon, Royal Victoria Hospital, Montreal, Canada. Third Edition. Published in St. Louis by C. V. Mosby Company, 1959. Price $17.00.

The third edition of this classical textbook continues the tradition of the past. The editing, as before, is excellent, the book is readable throughout, and the third edition has been brought very much up to date.

There is an excellent new chapter on pediatric surgery, and the fact that it was written by contributors from the Montreal Children's Hospital, denotes a change from the policy of the past, restricting contributors to the staff of the Royal Victoria Hospital and McGill University.

The chapters on surgery of the heart, great vessels and lungs have been entirely rewritten and up-dated.

There is much rewriting throughout the book. Additional color plates will also be found, bringing the total number to 108.

The 738 text illustrations are all excellent, and are presented in context.

The third edition of this book is worthy of a place in every surgeon's library.

The proctologist will find an excellent chapter on the colon and another on the rectum and anus, both by the same author (Harry S. Morton).

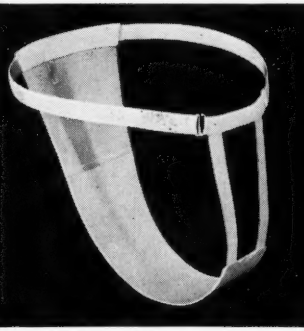
The book is an excellent reference work, as well as a fine teaching text.

CIBA FOUNDATION COLLOQUIA ON ENDOCRINOLOGY, volume 9, Internal Secretions of the Pancreas. G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Cecilia M. O'Connor, B.Sc., Editors. Published by Little, Brown and Company, Boston, Mass. Price $7.00.

This is the ninth volume of the series on endocrinology produced by the Ciba Foundation in London. The papers and discussions are informative and of interest to physicists,

—Continued on following page

AFTER ANORECTAL SURGERY



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BOOK REVIEWS

—Continued from preceding page

chemists, biochemists and biologists as well as to physicians principally concered with endocrinology.

Stress is placed on the modes in which hormones sometimes seem to mimic each other in action, as exemplified by growth hormone acting like insulin, insulin like growth hormone, etc. The point is made that the days when one hormone had one action and no other hormone had that action are long passed. Overlapping of actions is the rule. Also, one hormone injected into the circulation of an animal may bring about liberation of a series of hormones which may in turn influence a series of enzymes.

The subject becomes more complicated as time passes, but this and the other volumes in the series help to dispel some of the obscurity enveloping these problems. Despite the unavoidable technicalities and sometimes esoteric abstruseness of the specialist's language, the reader receives a sense of participation in these informal and friendly conferences and discussions.

"TUMORS OF THE BLADDER" by David M. Wallace, OBE, MS, FRCS, Surgeon, St. Peter's, St. Paul's, St. Philip's Hospitals; Associate Urologist, Royal Marsden Hospital; Senior Lecturer, Institute of Urology, University of London. Published by the Williams & Wilkins Company, Baltimore. 1959. Price $11.50.

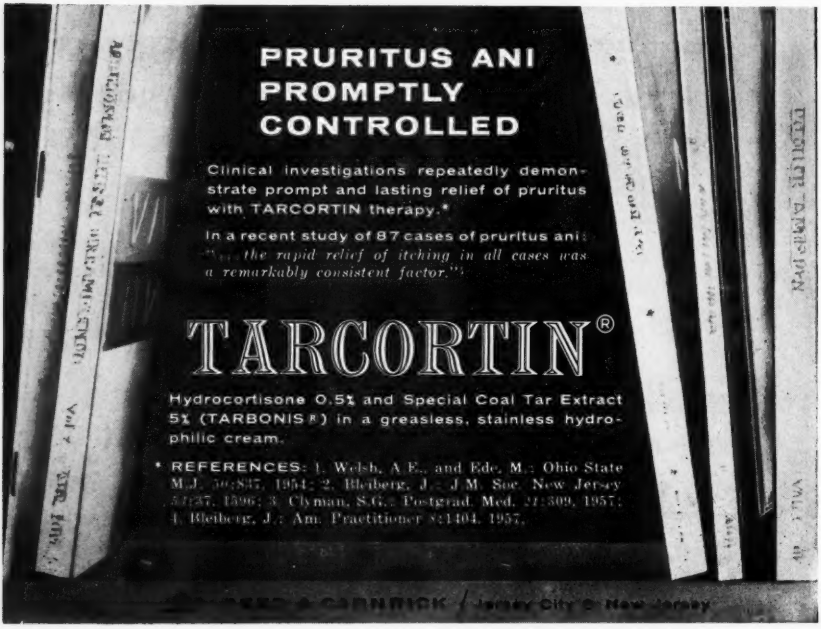
This is an English monograph, one of a series on neoplastic disease at various sites.

Tumors of the bladder may be produced, as the author points out, by a large number of chemical compounds. Indeed, some tumors referred to as spontaneous in origin may actually be due to an environmental exposure to particular chemicals. When certain chemicals are added to a normal diet bladder tumors are produced by the action of their metabolic end products excreted in the urine.

Inasmuch as occupational tumors of the bladder do appear, the importance of this chemical factor in England in terms of the N.H.S. 1946 Act is pointed out. Perhaps more recognition should be given to this possibility in the United States.

The contributors to this volume are all authorities in their respective fields. The writing is uniformly good, and the editing is excellent. Illustrations are very adequate.

This is a volume that every urologist will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want to have in his library, and one that will be useful to the general surgeon and the radiotherapist as well.

"A SHORT PRACTICE OF SURGERY" by Hamilton Bailey, F.R.C.S. (Eng.), F.A.C.S., F.I.C.S., F.R.S.E., Emeritus Surgeon, Royal Northern Hospital, London; Surgeon, Metropolitan Ear, Nose and Throat Hospital and St. Vincent's Clinic; Consulting Surgeon, Italian Hospital; Formerly Assistant Surgeon, Liverpool Royal Infirmary; Hunterian Professor of Surgery, Royal College of Surgeons. Sometime External Examiner, University of Bristol. McNeill Love, M.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S., F.I.C.S., Emeritus Surgeon, Royal Northern Hospital; Consulting Surgeon, Mildmay Mission Hospital, Metropolitan Hospital, City of London, Maternity Hospital, and Potters Bar Hospital; Associate for General Surgery, West End Hospital for Nervous Diseases; Late Member of Council and Chairman Court of Examiners, Erasmus Wilson Demonstrator and Hunterian Professor, Royal College of Surgeons. With Chapters by John Charmley, F.R.C.S. (Eng.), Orthopedic Surgeon, Manchester Royal Infirmary; Park Hospital, Davyhulme; Wrightington Hospital, nr. Wigan; Lecturer in Orthopedic Surgery, University of Manchester, etc. William P. Cleland, M.R.C.P. (London), F.R.C.S. (England), Surgeon, Brompton Chest Hospital; Thoracic Surgeon, King's College Hospital and Postgraduate Medical School, London; Consulting Thoracic Surgeon to the Royal Navy. Geoffrey Knight, F.R.C.S. (England), Senior Surgeon, West End Hospital for Neurology and Neuro-surgery; Neurological Surgeon, Postgraduate Medical School, London, and Queen Elizabeth Hospital for Children; Surgeon in charge S. E. Metropolitan Regional Neurosurgical Centre. Eleventh Edition. Published in 1959 by J. B. Lippincott Company, Philadelphia. Price $18.00.

There are two new chapters in the eleventh edition, and both are excellent. Considering the original size of the book, and the new chapters now added, the term "short" seems out of place. The book actually contains 1,350 text pages.

The new material deals with Fluid Balance and Radiotherapy. Some of the other material has been extensively rewritten, and there is new material on the pathology of breast disease. Endocrine therapy of prostate and breast is also discussed.

The newer developments of surgery during the three years between the tenth and eleventh edition are incorporated in the text. The book is recommended for its writing, careful editing, and its illustrations.

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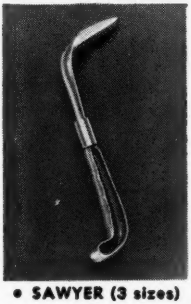

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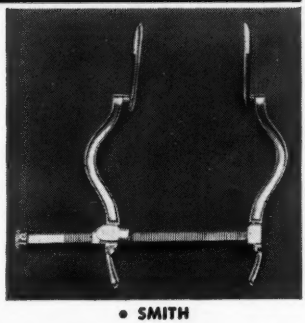

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Borcherdt Co. 178
Bristol Laboratories 166, 167
Burroughs Wellcome & Co., Inc. 211
Burton, Parsons & Co. .....Cover 3
Desitin Chemical Co. 160
Dittmar-Penn Corp. 215
Dome Chemicals, Inc. 168
Eaton Laboratories 175
Fuller Pharmaceutical Co.....162, 174, 213
Geigy Pharmaceuticals 164
Johnson & Johnson 176
Kirk Co., C. F. 172
Lloyd Brothers 177
Massillon Rubber Co., The 218
Merck Sharp & Dohme, Division of Merck & Co. Inc. 158
Pitman-Moore Co. 180
Reed & Carnrick 214
Searle & Co., G. D. .....Cover 2
Squibb & Sons, E. R., Division of Olin-Mathieson Chemical Corp. 155
Stuart Co., The 170, 171
U. S. Vitamin Corp. 217
Warner-Chilcott Laboratories 179
Welch Allyn, Inc. 161
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The Academy is grateful to C. J. Birtcher, President of the Birtcher Corporation, for his generous support of the International Academy of Proctology.